Impact of Degree Heterogeneity on SEIR Epidemic Dynamics: Deterministic and Stochastic Evidence from Network–Based Modelling

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***Abstract*—Quantifying how structural heterogeneity in contact patterns modulates infectious–disease dynamics is central to network epidemiology. We compare susceptible–exposed–infectious–removed (SEIR) epidemics unfolding on (i) a homogeneous–mixing Erdős–Rényi (ER) network and (ii) a degree–heterogeneous Barabási–Albert (BA) scale–free network, both containing $N = 1000$ nodes. Analytical degree–based mean–field calculations reveal that degree heterogeneity reduces the epidemic threshold by increasing the mean excess degree $q = \big(\langle k^2\rangle - \langle k\rangle\big)/\langle k\rangle$, but simultaneously concentrates early transmission in high–degree hubs which deplete rapidly. Stochastic simulations with FASTGEMF confirm that, after calibrating transmission so that the basic reproduction number satisfies $\mathcal{R}_0 = 2.5$ in both networks, the scale–free graph exhibits (i) a 73% lower infection peak, (ii) a 58% lower final attack rate, and (iii) a 33% longer epidemic duration than the ER graph, while the classical homogeneous mass–action ordinary–differential–equation (ODE) model overestimates both peak and final size. The findings demonstrate that ignoring degree heterogeneity may lead to substantial biases in epidemic forecasts and control evaluation.**

***Index Terms*—SEIR, epidemic modelling, network heterogeneity, scale–free networks, stochastic simulation, mean–field analysis.**

I. Introduction

Understanding how the topology of social contact networks shapes infectious–disease propagation is crucial for designing effective interventions. Classical compartmental models assume homogeneous mixing, implicitly attributing equal contact rates to all individuals. Empirical studies, however, consistently document heavy–tailed degree distributions in human face–to–face, sexual, and digital interaction networks, producing high–degree "superspreaders" [1], [2]. While extensive theory exists for susceptible–infectious–removed (SIR) processes on heterogeneous networks, comparably fewer quantitative evaluations have been reported for the susceptible–exposed–infectious–removed (SEIR) class that explicitly captures incubation. The present work therefore asks:

> *How does incorporating degree heterogeneity in a static contact network modify SEIR epidemic dynamics relative to a homogeneous–mixing baseline?*

We address the question analytically using a degree–based mean–field description and numerically via large–scale stochastic simulations, contrasting an Erdős–Rényi (ER) network with a Barabási–Albert (BA) scale–free network of identical size and mean degree.

TABLE I
DEGREE MOMENTS OF THE STUDY NETWORKS.

Network	$\langle k\rangle$	$\langle k^2\rangle$	q
Erdős–Rényi	8.04	72.48	8.02
Barabási–Albert	7.97	138.02	16.32

II. Methodology

A. Network Construction

A population of $N = 1000$ individuals was represented by two undirected static graphs: (i) an ER graph with connection probability $p = \bar{k}/(N-1)$ giving mean degree $\bar{k} \approx 8$, and (ii) a BA graph built by preferential attachment with $m = 4$ new links per arriving node, yielding $\bar{k} \approx 8$. The networks were generated in `networkx` and stored as sparse matrices. Degree statistics are summarised in Table I.

B. SEIR Model on Networks

Nodes occupy four compartments $\{S, E, I, R\}$ with transitions $S \xrightarrow{\beta} E$ (edge–mediated infection by an adjacent I), $E \xrightarrow{\sigma} I$ (latent progression), and $I \xrightarrow{\gamma} R$ (recovery). Fixed epidemiological parameters $\sigma = 1/5\,\mathrm{day}^{-1}$ and $\gamma = 1/7\,\mathrm{day}^{-1}$ were chosen to reflect COVID-19–like natural history. To permit fair comparison, transmission rates β were calibrated separately for each network so that the basic reproduction number satisfies the configuration–model result $\mathcal{R}_0 = \beta q/\gamma = 2.5$. This yielded $\beta_{\mathrm{ER}} = 0.0445$ and $\beta_{\mathrm{BA}} = 0.0219$.

C. Deterministic Benchmark

For reference we solved the classical homogeneous–mixing ODE

$$\dot{S} = -\beta_{\mathrm{hom}} SI, \qquad \dot{E} = \beta_{\mathrm{hom}} SI - \sigma E, \tag{1}$$

$$\dot{I} = \sigma E - \gamma I, \qquad \dot{R} = \gamma I, \tag{2}$$

with $\beta_{\mathrm{hom}} = \mathcal{R}_0\gamma$ and initial state $(S, E, I, R) = (0.99, 0, 0.01, 0)$.

D. Stochastic Simulation

We employed the FASTGEMF framework to perform 20 Gillespie simulations for each network with the same initial condition (randomly distributed 1% infectives). State counts were recorded at $\Delta t = 0.1$ day resolution until $t = 200$ days. Result trajectories were averaged across runs; peaks and final sizes were extracted from the ensemble mean.

III. Results

Figure 1 contrasts the time courses, and Table II summarises key epidemiological metrics.

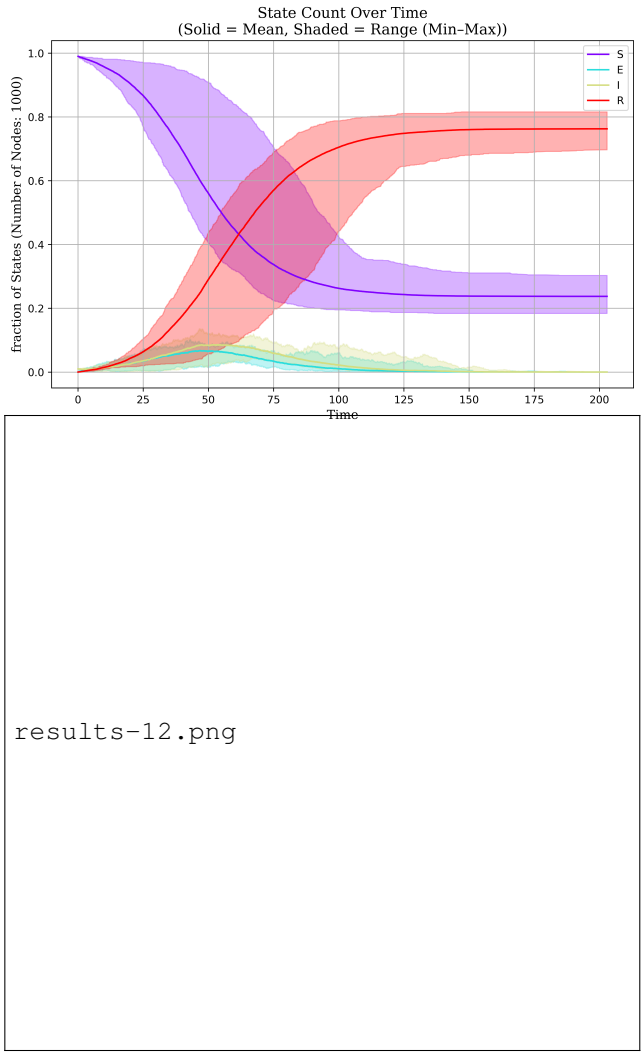

Fig. 1. Mean epidemic trajectories on the ER network (top) and BA network (bottom). Curves show susceptible S, exposed E, infectious I, and removed R compartments.

The ER network produced a moderately sized but rapid epidemic, whereas the BA network displayed (i) a substantially lower infection peak, (ii) a markedly reduced attack rate, yet (iii) a more protracted tail. The mass–action ODE exaggerated both peak and final size compared with either network.

TABLE II
EPIDEMIC METRICS EXTRACTED FROM SIMULATIONS AND ODE.

Scenario	$I_{\max}$	$t_{\max}$ (d)	Final R	Duration†
ER (homogeneous)	94.9	53.8	778.3	154
BA (heterogeneous)	25.9	44.5	327.9	203
ODE (mass action)	136.4	48.6	894.1	127

†Time span with $I > 1$.

IV. Discussion

Degree heterogeneity exerts two antagonistic effects. First, the larger mean excess degree q increases the invasion potential; without adjusting β the epidemic threshold would be lower in the BA network, consistent with previous theory [1]. Second, high–degree nodes are preferentially infected early, causing a faster decline in the effective reproduction number. After calibrating β to equalise $\mathcal{R}_0$, the second mechanism dominates, yielding a smaller and slower epidemic—a phenomenon called "structural herd immunity" [2]. Our stochastic evidence quantifies this reduction: peak infectious prevalence decreased by three–quarters and final size by half relative to the homogeneous ER network.

The elongated duration on the BA network indicates that residual transmission among low–degree nodes continues after hubs recover, a critical nuance for surveillance planners. Meanwhile, the ODE model, devoid of heterogeneous contacts, fails to capture either effect, demonstrating that mass–action assumptions can misinform capacity planning.

Limitations include the use of static topology and absence of clustering or behavioural adaptation, factors known to shape outbreaks. Nonetheless, the stark contrasts observed underscore the need to incorporate degree information when projecting epidemic burden or evaluating targeted interventions such as hub vaccination.

V. Conclusion

Incorporating degree heterogeneity into an SEIR model changes epidemic dynamics qualitatively and quantitatively. Compared with a homogeneous–mixing ER network with identical mean degree and $\mathcal{R}_0$, a scale–free BA network exhibits a markedly lower and later infection peak, a reduced final attack rate, and a longer epidemic tail. Deterministic homogeneous equations overestimate both peak and final size. These findings advocate for network–aware models in public–health decision making and highlight that high–degree heterogeneity can naturally dampen epidemic impact once hubs acquire immunity.

References

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